4. Swerdlow AJ, Higgins CD, Smith P, et al. Myocardial infarction mortality risk after treatment for Hodgkin disease: a collaborative British cohort study. *J Natl Cancer Inst.* 2007;99:206-214.
5. Castellino SM, Geiger AM, Mertens AC, et al. Morbidity and mortality in long-term survivors of Hodgkin lymphoma: a report from the Childhood Cancer Survivor Study. *Blood*. 2011;117:1806-1816.

Electrophysiology

Nonischemic Dilated Cardiomyopathy

Norman E. Lepor, MD, FACC, FAHA, FSCAI,[1] Gerald Pohost, MD, FAHA, FACC,[2] Donna Gallik, MD, FACC, FHRS[3]

[1]Westside Medical Associates of Los Angeles and Geffen School of Medicine-UCLA Cedars-Sinai Heart Institute, Los Angeles, CA; [2]Keck School of Medicine, University of Southern California, Los Angeles, CA, Loma Linda University, Loma Linda, CA, Westside Medical Imaging, Beverly Hills, CA; [3]Cedars-Sinai Heart Institute, Los Angeles, CA
[Rev Cardiovasc Med. 2014;15(1):73 doi: 10.3909/ricm0711]

Association of Fibrosis With Mortality and Sudden Cardiac Death in Patients With Nonischemic Dilated Cardiomyopathy

Gulati A, Jabbour A, Ismail TF, et al.
JAMA. 2013;309:896-908.

Identifying patients with nonischemic dilated cardiomyopathy (NIDC) who will benefit from internal cardioverter-defibrillators (ICDs) remains a major challenge in clinical electrophysiology. Being able to identify the patient at higher risk of sudden cardiac death (SCD) has major public health implications due to the relatively common prevalence of NIDC (1 in 2500 adults), the cost of ICD placement, inherent ICD implant risks, and the risk of inappropriate shocks.[1,2] Current American College of Cardiology/American Heart Association guidelines state that ICD therapy is indicated for primary prevention of SCD in patients with NIDC who have an ejection fraction $\leq$ 35% and who are in New York Heart Association functional class II or III.[3] However, the rate of appropriate discharge in patients with ICDs and left ventricular ejection fractions (LVEFs) $<$ 35% is only 5.1% per year, limiting the utility of this guideline-based approach.[4]

Gulati and coworkers evaluated the hypothesis that the presence of fibrosis within the left ventricular wall would add prognostic accuracy to LVEF in assessing the risk of SCD in patients with NIDC. They performed a prospective, longitudinal evaluation of 472 patients with NIDC referred to a single center in England between 2000 and 2008. Cardiovascular magnetic resonance imaging with late gadolinium enhancement (LGE-CMR) was used to determine the presence or absence of ventricular midwall fibrosis (MWF). The presence of LGE correlates histologically with the presence of fibrosis. In this study, the presence, location, and extent of fibrosis were assessed. The predefined endpoint was all-cause mortality with a predefined secondary endpoint being a composite of cardiovascular mortality, heart failure, stroke, or thromboembolic event. Two other prespecified endpoints included an arrhythmic composite of SCD or aborted SCD, and a heart failure endpoint of heart failure death, unplanned heart failure hospitalization, or cardiac transplantation.

Patients with MWF had an all-cause mortality of 26.8% compared with only 10.6% in those without MWF (hazard ratio = 2.96; $P < .01$). After a multivariate analysis, the presence of MWF remained a highly significant risk factor for total mortality and, to a lesser extent, the percentage extent of MWF. The secondary arrhythmic composite endpoint (sudden death, aborted sudden death, appropriate ICD shock, ventricular tachycardia, or sustained ventricular tachycardia) also was significantly higher in the patients with fibrosis (29.6% vs 7%; $P < .001$). Using the presence or absence of MWF in addition to LVEF, they found an additional 18.5% of patients who would have their SCD risk upgraded and undergo ICD implant and 10.6% of patients who would have their risk downgraded and avoid ICD implant.

The results of this single-center study show in dramatic fashion the potential utility of LGE-CMR for assisting with risk assessment of SCD and subsequent ICD implant. The results of this single-center study await confirmation by larger multicenter trials before generalized use can be recommended. ■

References

1. Jeffries JL, Towbin JA. Dilated cardiomyopathy. *Lancet*. 2010;375:752-762.
2. Epstein AE, DiMarco JP, Ellenbogen KA, et al. ACC/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices) developed in collaboration with the American Association for Thoracic Surgery and Society of Thoracic Surgeons. *J Am Coll Cardiol*. 2008;51:e1-e62.
3. Hunt SA, Abraham WT, Chin MH, et al. 2009 focused update incorporated into the ACC/AHA 2005 Guidelines for the Diagnosis and Management of Heart Failure in Adults: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines: developed in collaboration with the International Society for Heart and Lung Transplantation. *Circulation*. 2009;119:e391-e479.
4. Brady GH, Lee KL, Mark DB, et al; Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) Investigators. Amiodarone or an implantable cardioverter-defibrillator for congestive heart failure. *N Engl J Med*. 2005;352:225-237.